HYPOXIC-ISCHAEMIC BRAIN INJURY SECONDARY TO CARDIAC ARREST FROM SPINAL ANAESTHESIA: A CASE REPORT

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ABSTRACT

BACKGROUND: *Hypoxic-ischaemic brain injury is usually due to cardiac arrest or severe hypotension. The clinical manifestation and outcome will depend on the severity of the initial insult, the promptness and effectiveness of the resuscitation, and the post resuscitation management in the intensive care unit.*

OBJECTIVE: *To highlight the need for vigilance and preparedness in every case of spinal anaesthesia.*

CASE REPORT: *Patient was a 32years old housewife who was booked for emergency lower segment caesarean section on account of transverse lie in labour at term. She had no co-morbidities. A subarachnoid block was established with a 25G Quinke needle through L3 – L4 intervertebral space using 2ml of 0.5% heavy bupivacaine, after preloading, she was then wedged in a left lateral tilt. Five minutes after this procedure, and before the commencement of surgery, the patient had a cardiac arrest. She was resuscitated, intubated, ventilated, and delivered of a live asphyxiated baby. She was then transferred to the intensive care unit, where she eventually made a full recovery.*

CONCLUSION: *While hypoxic-ischaemic brain injury is not commonly associated with spinal anaesthesia, vigilance, and prompt and aggressive management is key to a favourable outcome.*

Keyword: *spinal anaesthesia, hypotension, cardiac arrest, hypoxic-ischaemic brain injury*

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INTRODUCTION

As one of the regional techniques in anaesthesia, spinal anaesthesia (subarachnoid block) is a safe and effective option to general anaesthesia especially when the surgical site is below the umbilicus. It involves the introduction of a local anaesthetic agent, with or without an opioid, into the subarachnoid space. Subarachnoid block produces intense sensory and motor blockade as well as sympathetic blockade. The sympathetic block produced by this anaesthetic technique can cause severe hypotension in the absence of preloading of the circulatory system with crystalloids or colloids, or the use of a vasopressor[1].

Hypoxic-ischaemic brain injury has been described as a complex constellation of pathophysiological and molecular injuries to the brain induced by hypoxia, ischaemia, cytotoxicity, or combinations of these conditions[2]. The typical causes include cardiac arrest, respiratory arrest, near drowning, near-hanging, and other forms of incomplete suffocation, carbon monoxide and other poisonous gas exposures, and perinatal asphyxia. These expose the entire brain to potential injurious reductions of oxygen (hypoxia) and/or diminished blood supply (ischaemia)[3].

The terms “anoxic brain injury” and “anoxic brain damage” are the most commonly used clinical and research descriptors of this condition, and are generally used as synonyms for hypoxic-ischaemic brain injury. These terms however overstate the severity of one pathophysiologic contributor to injury; decreased delivery of oxygen to the brain, most accurately described as hypoxia rather than anoxia, and ignore entirely the often concurrent and more injurious decrease in

perfusion of the brain (ischaemia) [3]. True anoxia (complete absence of oxygen in the blood) is a rare and debatably survivable event.

CASE REPORT

Patient was a 32years old housewife who was booked for emergency lower segment caesarean section on account of transverse lie in labour at term. She was not a known hypertensive, diabetic or asthmatic. She was also not a known epileptic and had no history of allergy. The patient had no previous surgical or anaesthetic experience. Family and social history were not contributory. Examination revealed a young woman who was in obvious painful distress, afebrile, not pale, moderately dehydrated, tachypneic but not cyanosed. Pulse rate was 105beats /minute and blood pressure was 120/70mmHg. The chest was clinically clear. Haematocrit was 32% and urinalysis showed normal findings. She was Mallampati class 2 and ASA class 1E. Spinal anaesthesia was the planned anaesthetic technique. A consent form was signed and one pint of whole blood was ordered grouped and cross-matched.

In the theatre, the anaesthetic machine was checked, appropriate sizes of endotracheal tubes and laryngoscope blades were made available, and a multiparameter monitor connected to patient for baseline vital signs. Baseline pulse rate was 110beats/minute and blood pressure was 120/70mmHg.A peripheral venous access was established on the left forearm and a urinary catheter was passed. The patient was then preloaded with 1000ml of 0.9% saline. Both ranitidine 50mg and metoclopramide 10mg injections were administered intravenously.

A subarachnoid block was established using a size 25G Quinke needle, with the patient in the sitting position, with 2ml of 0.5% heavy bupivacaine through L3-L4 intervertebral space. Patient was then placed in the supine position with slight head up, and wedged in a left lateral tilt. Immediate post-spinal pulse and blood pressure checks were a pulse rate of 70beats/minute and blood pressure of 100/70mmHg. About 5 minutes after establishing subarachnoid block, while the operation field was being cleaned and draped, the patient complained of tightness around her chest. The level of block was then at T6, the oxygen saturation was 98%, the pulse rate was 50beats/minutes and the blood pressure was 90/50mmHg. Intravenous atropine 3mg and ephedrine 6mg were given and an additional litre of 0.9% saline rushed in. Oxygen by facemask was commenced. Within a minute of these measures there was absence of peripheral pulsation and cessation of cardiac activity. A diagnosis of cardiac arrest was made and chest compression was started. Adrenaline 1mg was given intravenously.

Two attempts at intubating the patient were unsuccessful. A third attempt by a consultant anaesthetist was successful. The vital signs of patient rapid improved as intermittent positive pressure ventilation (IPPV) with 100% oxygen was commenced. The duration of arrest was about six minutes. The patient was delivered of a live, moderately asphyxiated, female baby. The baby was successfully resuscitated. A second peripheral venous access was opened and maintained with 0.9% saline. Patient was maintained with 0.4% halothane in 100% oxygen. She was also given pentazocine 45mg, paracetamol 1000mg, ceftriaxone 1000mg, and metronidazole 500mg intravenously. Mean arterial blood pressure was between 60 – 70mmHg, oxygen saturation was 100% and pulse rate was 80beats/minutes. Electrocardiographic and end tidal carbon dioxide monitoring were not available in the theatre as they were not parameters measured by the multiparameter monitor used. Surgery lasted approximately 45minutesand the estimated blood loss was about 500ml.She received a total of 4L of 0.9% normal saline.

Just before the end of surgery, patient had an episode of convulsion. This was initially subtle but very quickly became generalised tonic-clonic. This was abated with 250mg intravenous sodium thiopentone. A repeat episode of convulsion necessitated paralysis

with iv atracurium 25mg. A clinical diagnosis of hypoxic-ischaemic brain injury was made on account of the intraoperative events.

Postoperatively, the patient was transferred to the intensive care unit, with the endotracheal tube in-situ and a bag-valve-mask, and connected to a ventilator. She was sedated with infusion midazolam 0.05mg/kg/hr and had iv pentazocine 30mg 6hrly. Intravenous antibiotics were continued with close monitoring of vital signs including the use of an end-tidal carbon dioxide monitor.

There were no further episodes of convulsions, even during intervals of no sedation. She was waned off the ventilator after about 36hours. Serum electrolytes and end tidal carbon dioxide values were within normal. Arterial blood gas analysis and a CT-scan were not done because of lack of availability. She was discharge to the ward 72hrs after admission into the ICU for follow-up by the neurologist. She had no further episodes of convulsion during her stay in the ward.

DISCUSSION

Cardiac arrest is the commonest cause of hypoxic-ischaemic brain injury. It is usually due to arrhythmias, but it may also follow respiratory arrest or hypotension due to shock or hypovolaemia. The initial arrhythmia is ventricular tachycardia or ventricular fibrillation in approximately 50% and asystole or pulseless electrical activity in the reminder. However, the longer the rescue time, the more the risk of asystole[4]. There have been some debates regarding the aetiology of the arrests seen during spinal anaesthesia. The evidence for a respiratory aetiology for these arrests is sparse. Spinal anaesthesia sensory levels up to T4 do not lead to hypoventilation, but are associated with mild hyperventilation[5]. Also, sedation and hypoxaemia, as primary causes of cardiac arrest in spinal anaesthesia have been discarded as these arrests usually occur in oxygen saturation settings of 95 – 100%[6]. The patient we reported was not sedated and had an oxygen saturation of 98% when she developed cardiac arrest.

Evidence for a circulatory aetiology regarding these arrests is now more widely accepted. This comes from physiological studies using healthy volunteers who have experience bradycardia and cardiac arrests in settings that mimic the effect of spinal anaesthesia[7]. Most of these effects are directly or indirectly related to the blockade of sympathetic efferents during spinal anaesthesia. The extent of sympathetic blockade is often two to six levels higher than the sensory level, so that a patient with sensory block T4 may have completely blocked cardiac accelerator fibres that originate from T1 to T4[8]. These can lead to a variety of bradyarrhythmias.

A more important effect of the inhibition of the sympathetic efferents during spinal or epidural anaesthesia is a significant decrease in venous return to the heart. Baron et al found that cardiac vagal tone is enhanced primarily through reduced venous return[9].The effect of spinal anaesthesia on venous return can be profound. Reductions in the right atrial pressure of 36% after low spinal levels (below T4) and by 53%after higher levels of blockade have been reported[10].

These decreases in preload may initiate reflexes that cause severe bradycardia. Three such reflexes have been suggested. The first involves the pacemaker stretch. The rate of firing of these cells within the myocardium is proportional to the degree of stretch. Decreased venous return results in decreased stretch and a slower heart rate. The second reflex may be attributable to the firing of low-pressure baroreceptors in the right atrium and vena cava. The third is a paradoxical Bezold-Jarisch reflex, in which mechanoreceptors in the left ventricle are stimulated and cause bradycardia[11].Since a high degree of cardiac vagal activity can occur during spinal anaesthesia, patients with strong resting vagal tone should be at increased risk for cardiac arrest during spinal anaesthesia[8]. In particular, spinal anesthesia has been associated with

progression of first-degree heart block to second-degree heart block[12]. Complete heart block and cardiac arrest may simply represent the most severe vagal-induced bradyarrhythmia associated with spinal anesthesia[13].

Carpenter et al reported that a baseline pulse of<60beats/minutes was associated with a fivefold increase in the odds of developing moderate bradycardia (heart rate of between 50 – 55 beats/min) during spinal anesthesia. Typically, young patients have strong vagal tone, and they reported that ASA physical status I patients have a threefold increased risk for developing moderate bradycardia during spinal anaesthesia. Current therapy with **beta**-blockers or block height above T6 was also important risk factors for bradycardia identified in the same study. Other researchers have reported that patients who are above 50 years old and patients with first-degree heart block are also at increased risk for moderate bradycardia during spinal anaesthesia[14].

These factors are known to increase the risk of moderate bradycardia during spinal anesthesia and could help identify patients at risk for cardiac arrest during spinal anesthesia. The presence of a single risk factor does not make it certain that a patient will experience severe bradycardia or cardiac arrest. However, when two or more of the factors are present, the patient may be considered high-risk for bradycardia and cardiac arrest during spinal anaesthesia[14].Our patient was ASA 1 and was below 50 years.

Pregnancy is associated with changes in autonomic control and, at term, heart rates of 90–95beats/minutes are typical; this may be attributable to decreased parasympathetic tone during pregnancy[15]. If vagal predominance plays a key role in the cardiac arrests that occur during spinal or epidural anaesthesia, then the weaker vagal tone associated with pregnancy may decrease this risk. This was not found to be the case in the patient presented.

When spinal anaesthesia has been selected for a patient, maintaining adequate preload is key to decreasing the risk of bradycardia and cardiac arrest during the anaesthetic procedure. Decreases in preload can occur so quickly with altering position and other common perioperative events that there may not be time to give sufficient volumes of fluid over several minutes. When an abrupt decrease in preload is suspected, placing the patient in the head-down position and rapidly infusing IV fluids can be helpful[7]. Treating mild bradycardia (heart rate of between 56 - 60beats/minutes) during spinal anaesthesia may be appropriate especially if the patient has multiple risk factors. Atropine is recommended to treat bradycardia during spinal anaesthesia because glycopyrrolate is ineffective in this setting[16].

Brown et al reported three cardiac arrests during a period when 10,080 spinal anaesthetics were performed without any episode of cardiac arrest resulting in neurologic injury[17]. This was attributed to vigilance and their "willingness to utilize IV atropine (0.4–0.6 mg), ephedrine (25–50 mg), and adrenaline (0.2– 0.3 mg) in stepwise escalation of therapy when bradycardia develops following spinal anaesthesia".This was the protocol employed in the case presentation.

Unfortunately, not all arrests that occur during spinal anesthesia are successfully treated, and fatal arrests still occur in healthy patients as was nearly experienced in our patient. The vasodilation caused by spinal anaesthesia can make cardiopulmonary resuscitation ineffective. Successful resuscitation requires a coronary perfusion pressure gradient of 15 to 20 mmHg and during spinal anaesthesia this may require adrenaline 0.01 to 0.1 mg/kg[18].The difficulty experienced in maintaining a patent airway in our patient also negatively affected the process of resuscitation.

The supine hypotensive syndrome of pregnancy is induced by compression of the inferior caval vein by the gravid uterus. Since uterine blood flow is dependent on perfusion pressure, hypotension results in

reduced uterine blood flow, with a potential compromise in fetal oxygenation. Thus the hypotensive effect of spinal anaesthesia per se may thus be aggravated in a significant number of term parturients. Studies have shown that colloid is superior to crystalloid in preventing post-spinal hypotension for elective cesarean delivery[19].Available resources limited us to the use of crystalloids in the management of the case reported. Colloid is a more effective volume expander because it stays longer in the intravascular compartment. The common clinical practice of volume expansion with crystalloid is not uniformly effective in reducing the incidence of maternal hypotension after spinal anesthesia for cesarean delivery[19]. Also during pregnancy, venous blood volume increases in the lower extremities, particularly after the 30th week of gestation, with spinal anaesthesia further increasing the volume of blood in the legs by induction of a pharmacologic sympathectomy[20].

The clinical effects and consequences of hypoxic–ischaemic brain injury are highly variable. The best outcome is generally seen in patients who have had only a short period of impaired consciousness following cardiac arrest. Regaining purposeful motor movements and awareness with preserved memory within a few hours is usually associated with a good recovery and functional independence. However, the literature on prognostic factors and outcome is difficult to apply these days because it is largely based on studies before the modern techniques of post-resuscitation support, including the early use of sedation and ventilation, which now prevent a full assessment of conscious level and more or less any informative neurological examination in the immediate post-arrest period[14].

Seizures (event-related and recurrent), disturbances of sensorimotor function, and a broad array of cognitive, emotional, and behavioural disturbances are commonly consequences of this brain injury[21]. As many as one-third of individuals sustaining a hypoxic-ischaemic brain injury develop seizures in the immediate post-injury period, typically beginning within 24 hours of injury but occurring and recurring over the first two weeks thereafter[14].

Most post-hypoxic seizures usually are partial complex or myoclonic in character and occur intermittently. The occurrence of early seizures does not necessarily portend the development of post-hypoxic epilepsy or persistent post-hypoxic myoclonus, nor does it invariably predict poor neurological or functional outcome. However, the post-hypoxic status epilepticus (SE) or the myoclonic SE is associated, almost invariably, with a fatal outcome from hypoxic-ischaemic brain injury[22].

In summary, a poor prognosis is likely, in the absence of sedating medication, which these days is unusual, if the corneal and pupillary light reflexes and motor responses are absent at 24 hours and 72 hours[14]. These reflexes were present in the patient presented.

CONCLUSION

This case report highlights the need for vigilance even when a subarachnoid block may seem straightforward. The availability of emergency drugs and airway management devices cannot be overemphasised.

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